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Racial Differences in the Significance of Coronary Calcium in Asymptomatic Black and White Subjects With Coronary Risk Factors

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OBJECTIVES To compare the significance of a specific feature of coronary atherosclerosis—coronary calcium—in asymptomatic black and white subjects with coronary risk factors.

BACKGROUND The natural history and clinical evolution of coronary atherosclerosis differs between blacks and whites. Differences in the underlying pathobiology of atherosclerosis may be one determinant of the ethnic variability in the clinical manifestation of coronary atherosclerosis.

METHODS In 1,375 high-risk but asymptomatic subjects (93 blacks [6.8%] and 1,282 whites [93.2%]) with at least one risk factor but no prior evidence of coronary disease, we assessed coronary risk factors, calculated Framingham risk of a coronary event and evaluated coronary calcium with digital subtraction fluoroscopy. We then followed these subjects clinically for 70 ± 13 months, noting the occurrence of the following coronary events: death due to coronary heart disease (CHD); myocardial infarction (MI); angina pectoris; and performance of coronary bypass or angioplasty.

RESULTS Risk factor profiles were similar in black and white subjects (6-year Framingham risk 15 ± 7% in blacks, 14 ± 8% in whites [NS]). Coronary calcium was present in 59.9% of white subjects but only 35.5% of black subjects ($p = 0.0001$). Nevertheless, after 70 months of follow-up, more blacks than whites (22 blacks [23.7%] vs. 190 whites [14.8%]; $p = 0.04$) suffered one of the following end points: CHD death, MI, angina or revascularization. The age, gender and coronary risk-adjusted odds ratio of black race for at least one event was 2.16 (95% CI 1.34 to 3.48).

CONCLUSIONS Despite having a lowered prevalence of coronary calcium than high risk whites, high risk blacks suffer more CHD events. Coronary calcium therefore does not carry the same pathobiologic significance in blacks that it does in whites, consistent with the concept that there are specific racial differences in the natural history of CHD and its evolution into clinically manifest events. (J Am Coll Cardiol 1999;34:787–94) © 1999 by the American College of Cardiology

Blacks suffer an inordinately high degree of adverse cardiovascular events (1–3), posing an important but unsolved public health dilemma. While both underutilization (4) and diminished access to health care services (5) appear to significantly contribute, there are persistent suggestions that there may be ethnic differences in the pathobiology of atherosclerosis that may affect the clinical expression of the disease.

Ethnic differences in risk factors appear early in childhood (6,7) and may impact subsequent racial variation in clinical expression of the underlying substrate. For example, data from several sources indicate that smoking has a disproportionately lethal effect on blacks (8–10). The Atherosclerosis Research in Communities (ARIC) study (11) and other reports (12–14) have implicated socioeconomic status as a contributor to cardiovascular morbidity and mortality. However, data from the Meharry-Hopkins Study in black and white physicians, presumably of comparable socioeconomic status, indicate that systolic blood pressure has a higher adjusted odds ratio for coronary events in black physicians but serum cholesterol is more deleterious in white physicians (15). Recent 16 year follow-up data from the Coronary Artery Surgery Study (CASS) indicate that even after adjusting for all clinical and treatment variables, black race was a strong predictor of mortality in both the surgical and medical groups (8). Collectively, these data are consistent with the concept that there are ethnic differences

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Abbreviations and Acronyms

CHD = coronary heart disease
CI = confidence interval
ECG = electrocardiographic
HDL = high density lipoprotein
LVH = left ventricular hypertrophy
MI = myocardial infarction
OR = odds ratio

in the pathobiology of atherosclerosis that affect its eventual clinical manifestations.

The pathobiologic substrates underlying these ethnic differences are unknown, but recent studies suggest the distinct possibility that coronary calcium—a specific and widely prevalent feature of coronary plaque—does not carry the same significance in blacks that it does in whites. For example, black subjects have a lower incidence of coronary calcification compared to whites, despite having a similar risk factor profile (16,17) and thus a roughly comparable risk of coronary heart disease (CHD) events. Furthermore, even after adjusting for the effects of all known determinants of coronary calcium (age, gender, and $1\alpha,25(OH)_2D_3$ [16,18]) as well as all other coronary risk factors, high risk asymptomatic blacks still have less coronary calcium than high risk asymptomatic whites (16). Based on available data on the prognostic significance of coronary calcium (19–24), this lesser amount of calcium in blacks should predict fewer subsequent events.

On the contrary, we report that although blacks have a lower prevalence of coronary calcium than whites, they nevertheless suffer greater numbers of coronary events, even after adjusting for the effects of risk factors. Therefore, coronary calcium is of different pathobiologic significance in black subjects with coronary risk factors compared to white subjects with similar risk factors.

METHODS

Subjects. All subjects gave written informed consent to voluntarily participate in this investigation and no subject was required to pay for testing. The study received approval from the Human Subjects Committee at our institution. Subjects were excluded if they had history or electrocardiographic evidence of a prior myocardial infarction (MI) or if they answered affirmatively to the first and two other questions from the following angina questionnaire: a) Do you suffer from chest discomfort? b) Does it last less than 15 minutes? c) Does the pain come on when you exert yourself? d) Is the pain relieved by rest or nitroglycerin? This definition of angina is similar to that used by Diamond and Forrester (25) and that used by the Framingham investigators (26).

A total of 1,461 subjects satisfied these enrollment criteria. Eighty six subjects from ethnic groups other than black or white (82 Asian Americans, 4 Native Americans) were excluded from analysis in this report. The remaining 1,375 subjects were either black (n = 93; 6.8%) or white (n = 1,282; 93.2%), were asymptomatic at the time of coronary calcium assessment, underwent risk factor evaluation at the time of coronary calcium screening, and were followed prospectively thereafter by the South Bay Heart Watch Clinic to determine coronary events. Details of recruitment, selection and risk factor evaluation methodology have been previously published (23,24,27).

Table 1. Coronary Heart Disease Risk Factors and Ethnicity

Risk Factor	Blacks	Whites	p Value
Age (yr)	60 ± 8	64 ± 8	0.0001
Male (%)	86	88	0.67
History of hypertension (%)	54	43	0.03
Systolic blood pressure (mm Hg)	150 ± 22	143 ± 18	0.002
History of diabetes (%)	27	17	0.01
Total cholesterol (mg/dl)	246 ± 47	241 ± 49	0.32
High-density lipoprotein cholesterol (mg/dl)	46 ± 12	44 ± 14	0.25
Currently smoking (%)	22	21	0.96
Any smoking history	77	73	0.30
Family history (%)	32	46	0.01
Left ventricular hypertrophy by electrocardiogram (%)	8.6	5.9	0.30
Body mass index (kg/m^2)	32 ± 5	30 ± 5	0.02
Six-year Framingham risk (%)	15 ± 7	14 ± 8	0.64

Entries are either mean values ± 1 SD or percentages. Comparisons use chi-square or Student's *t* test.

Our cohort was, by design, high-risk but asymptomatic, with no clinical or electrocardiographic (ECG) evidence of CHD, but all had >8% risk of a coronary heart disease event within six years, based on risk factor analysis and calculations utilizing the Framingham risk algorithm (26,28). All 1,375 subjects underwent evaluation of coronary calcium using digital subtraction fluoroscopy (23,24,27,29) and were evaluated annually thereafter.

Risk factor analysis. The following risk factors were assessed for each subject at the time of digital fluoroscopic coronary calcium assessment: age, gender, total serum cholesterol, high-density lipoprotein (HDL) cholesterol, systolic blood pressure, body mass index, history of hypertension, history of diabetes mellitus, history of smoking, family history of coronary heart disease, and left ventricular hypertrophy (LVH) by ECG (Romhilt-Estes LVH score) (Table 1). We also obtained information on current medication usage (Table 2). Details of risk factor assessment methodology are reported elsewhere (23,24,27).

Ethnicity. Ethnicity was determined by a clinical research nurse who questioned subjects regarding their ethnic ancestry and classified each subject as African American, Asian American, white, or Native American. Of the 1,461 subjects

Table 2. Medication Use by Race

Medication	Blacks	Whites	p*
Aspirin†	33%	43%	0.16
Beta-blocking agents	16%	9%	0.06
Other antihypertensive agents	58%	46%	0.06
Cholesterol-lowering medications	20%	26%	0.16

†Aspirin taken at least four times per week. *Two-tailed Fischer exact text. Entries are percentages. Comparisons use two-tailed chi-square.

enrolled, there were a total of 1,282 whites (87.7%), 93 blacks (6.4%), 82 Asian Americans (5.6%) and 4 Native Americans (0.3%). The proportion of minority subjects enrolled was roughly comparable to the ethnic compositions of the communities from which these subjects originated (17).

Digital subtraction fluoroscopy. We performed digital fluoroscopy in all subjects in the 60° left anterior oblique projection utilizing a Philips digital cinefluoroscopic x-ray imaging system. Further details of the image acquisition protocol have been reported elsewhere (17,23,24,27). Although there are no published head-to-head comparisons of digital fluoroscopy with the newer radiographic technology, electron beam computed tomography, we expect that the former technology will be somewhat less sensitive than the latter. All fluoroscopic images were assessed for the presence or absence of calcium in the distribution of each of the coronary arteries by two observers (RCD and WT), both blinded to risk factor information as well as to the other observer's interpretation. Interobserver differences in interpretation occurred in 7.7% of cases and were resolved by the two observers after review and consensus decision (30).

Follow-up. Subjects were assessed annually after enrollment, risk factor assessment and coronary calcium fluoroscopy, and then followed for 70 ± 13 months (successful follow-up was obtained in over 98% of subjects). The mean follow-up duration was 72 ± 13 months and 70 ± 13 months for blacks and whites, respectively (p = NS).

There were a total of six follow-up evaluations during the 70 ± 13 month follow-up period. Of the 93 black subjects in the cohort, 91 (97.8%) returned to the South Bay Heart Watch Clinic for at least one visit. Of the 1,282 white subjects, 1,248 (97.3%) returned to our clinic for at least one visit. The first two annual follow-up evaluations occurred principally in the South Bay Heart Watch Clinic, and the remaining four evaluations were largely by telephone.

At each follow-up visit evaluation, we assessed CHD status using review of medical records and transcriptions of conversations with families for infarctions, revascularizations and CHD deaths and the responses to the same angina questionnaire administered as on the initial visit (23,24,27). Three cardiologists met quarterly to decide by majority rule on all cases for which at least one member had diagnosed an MI (using criteria specified below, and previously reported [23,24,27]). Subjects unable or unwilling to return to the clinic were contacted by telephone by a research nurse and were either visited in their home by staff personnel or were mailed a questionnaire to obtain information on angina and hospital admissions.

Medical records were obtained for all but two hospital admissions (one black and one white subject). For subjects who died outside of a hospital, we obtained transcriptions of conversations with next of kin regarding circumstances and symptoms preceding death. Coronary heart disease end points were: 1) angina pectoris; 2) acute MI; 3) myocardial revascularization; and 4) CHD death. Evaluation of medical records to determine CHD end points was performed by a committee of three board-certified cardiologists without knowledge of coronary calcium results, ethnic origin or risk factor data. Evaluations by committee members were performed separately, and the entire committee would then meet periodically to decide by majority rule only those cases where there was not unanimous agreement among committee members whether or not an event had occurred. At follow-up, angina pectoris was defined as present by a score of 4 on the angina questionnaire that had been administered at screening. Thus, an increase in angina score from baseline of at least two was required in order to achieve this endpoint.

Coronary heart disease death was determined by the committee without knowledge of coronary calcium, ethnicity or other data by review of medical records (23,24,27). The committee also attributed each death either to CHD or non-CHD causes.

Statistical analysis. Two-tailed Fisher's exact test was used to evaluate ethnic differences in coronary calcification and CHD events. Either two-tailed Fisher's exact test or Student's *t* test was performed to compare differences in risk factors between ethnic groups. These tests were utilized to provide the most conservative method possible to test our hypotheses. Logistic regression was performed to evaluate the influence of risk factors and ethnicity on the occurrence of CHD events. Kaplan-Meier curves were compared using the Wilcoxon test. An alpha value <0.05 was considered significant.

RESULTS

Risk factors. Coronary heart disease risk factor data comparing black and white subjects are depicted in Table 1. Black subjects had a significantly higher systolic blood pressure (p = 0.002) and body mass index (p = 0.02), a higher incidence of diabetes mellitus (p = 0.01) and more frequent history of hypertension (p = 0.03) compared to white subjects. However, white subjects were significantly older (p = 0.0001) and had a greater incidence of positive family history of CHD compared to black subjects (p = 0.01). There were no significant ethnic differences in gender, total cholesterol, HDL cholesterol, history of smoking, current smoking status, LVH by ECG, or calculated Framingham risk of a coronary event in six years (Table 1).

Table 3. Comparison of the Topological Distributions of Coronary Calcium by Race

Number of Calcified Arteries	All Blacks (n = 93)	All Whites (n = 1,282)
None	64.5%	40.2%
One	21.5%	30.3%
Two	8.6%	18.0%
Three	5.4%	11.5%
	Blacks with Ca^{++} (n = 33)	**Whites with Ca^{++} (n = 768)**
One	60.6%	50.6%
Two	24.2%	30.1%
Three	15.2%	19.3%

In the top half of the table, numbers shown in the second column (blacks) represent percentages of the black cohort (n = 93). Numbers in the third column (whites) represent percentages of the white cohort (n = 1,282). In the bottom half of the table, the proportions shown in the second column are percentages of the black cohort who had at least some detectable calcium (n = 33). The percentages in the third column are percentages of those white subjects who had some detectable calcium (n = 768).

Table 4. Independent Predictors of the Presence of Any Coronary Calcium

Variable	Odds Ratio	95% CI
Age (per decade)	2.57	2.12, 3.12
Smoking	1.62	1.20, 2.20
Hypertension	1.46	1.15, 1.86
Family history	1.24	1.10, 1.39
Left ventricular hypertrophy by electrocardiogram	1.79	1.05, 3.05
Male gender	1.26	1.03, 1.53
Diabetes	1.19	1.01, 1.41
Total cholesterol (per 10 mg/dl)	1.03	1.01, 1.06
High-density lipoprotein cholesterol (per 10 mg/dl)	0.96	0.88, 1.05
Black race	0.47	0.29, 0.75
Body mass index (per 10 kg/m^2)	0.90	0.70, 1.17

Black race was a significant inverse predictor of coronary calcium presence. Age, smoking history, hypertension, family history, LVH, male gender, diabetes and total cholesterol were all significant independent predictors of coronary calcium.

There were also no significant ethnic differences in medication usage (Table 2), although there were trends towards more use of both beta blockers and other antihypertensive agents in black subjects ($p = 0.06$ for both comparisons).

Coronary calcium prevalence. The prevalence of any coronary calcium was 58.2% (n = 801) in the entire cohort. The prevalence of coronary calcium in black subjects was 35.5% (n = 33) and in white subjects was 59.9% (n = 768); this difference was highly significant ($p < 0.0001$).

Distribution of coronary calcium. Of the entire cohort, a total of 409 subjects (29.7%) had calcium in one coronary artery, 239 subjects (17.4%) had calcium in two coronary arteries and 153 subjects (11.1%) had calcium in all three coronary arteries.

Racial differences in the topographic distributions of coronary calcium are summarized in Table 3. Of the black cohort, 20 subjects (21.5%) had calcium in one coronary artery, 8 subjects (8.6%) had calcium in two coronary arteries and 5 (5.4%) had calcium in all three coronary arteries. Of the white cohort, 389 (30.3%) had calcium in the distribution of only one coronary vessel, 231 subjects (18.0%) had calcium in two coronary arteries, while 148 (11.5%) had calcium in all three coronary vessels.

Determinants of coronary calcium. Multivariate logistic regression analysis demonstrated black race to be a significant, independent and inverse predictor of the presence of coronary calcium (odds ratio [OR] 0.47; 95% confidence interval [CI], 0.29, 0.75). Age, male gender, smoking status, family history, diabetes mellitus, LVH, and serum total cholesterol were significant independent predictors of the presence of coronary calcium by digital fluoroscopy. These results are summarized in Table 4, along with the corresponding ORs and 95% CIs.

Coronary heart disease events. Despite having a lower prevalence of coronary calcium, more black subjects than white subjects suffered a CHD endpoint during the 70 month follow-up period. This difference was highly significant ($p = 0.001$), and is depicted in Figure 1, which shows event-free survival for blacks and whites as a function of time.

Table 5 shows ethnic differences in the occurrence of end points during the follow-up period. The occurrence of any end point (CHD death, nonfatal MI, new onset angina, or performance of a revascularization procedure) was more frequent in black subjects than in white subjects (22 blacks [23.7%]; 190 whites [14.8%]; $p = 0.04$). Blacks were also significantly more likely to suffer either CHD death, MI or new onset angina compared to white subjects (20 blacks [21.5%]; 162 whites [12.6%]; $p = 0.03$).

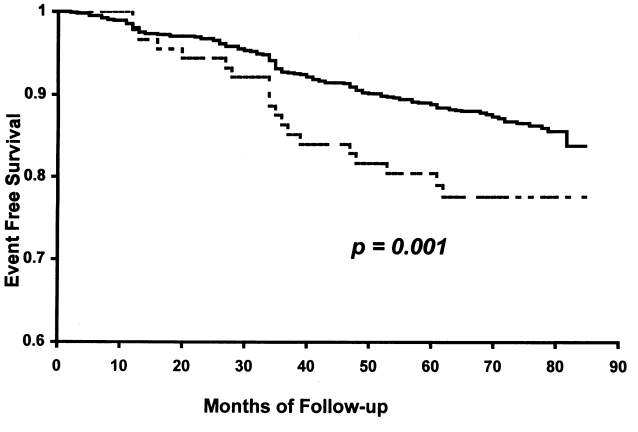

Figure 1. Kaplan-Meier event-free survival curves in black and white subjects during the 70 month follow-up period. Black subjects were significantly more likely than white subjects to suffer a coronary event ($p = 0.001$; Wilcoxon test).

Table 5. Coronary Events and Ethnic Origin at 70 Months of Follow-up

Event	Blacks*	Whites†	p Value
New-onset angina	15.1	7.6	0.02
MI	9.7	4.8	0.05
CHD death	4.3	2.9	0.35
Revascularization	4.3	5.9	0.65
Total deaths	11.8	9.0	0.35
CHD death or MI	11.8	6.7	0.09
CHD death, MI or angina	21.5	12.6	0.03
CHD death, MI angina, revascularization	23.7	14.8	0.04

*Entries represent percentages of black cohort (n = 93). †Entries represent percentages of white cohort (n = 1,282).

Blacks were significantly more likely to suffer new-onset angina pectoris than white subjects. Black subjects were also more likely to suffer one of the end points of CHD death, infarction or angina. In addition, blacks more frequently suffered any end point. There was a trend toward more hard coronary events (CHD death or infarction) in black subjects, but this trend did not reach statistical significance. There were no ethnic differences in CHD death rates, frequency of coronary revascularization procedures or total death rates from any cause.

CHD = coronary heart disease; MI = myocardial infarction.

Table 7. Independent Predictors of Any Coronary Event by Multivariate Logistic Regression, Along With Odds Ratios and 95% Confidence Intervals

Variable	Odds Ratio	95% CI
Black race	2.16	1.34, 3.48
Left ventricular hypertrophy	2.18	1.35, 3.52
History of diabetes	1.21	1.02, 1.43
Number of calcified vessels	1.16	1.01, 1.33
History of hypertension	1.34	1.02, 1.76
Total cholesterol (per 10 mg/dl)	1.03	1.00, 1.06
Family history of coronary heart disease	1.13	0.99, 1.29
Male gender	1.01	0.81, 1.25
Age (per decade)	1.09	0.89, 1.34
Smoking history	1.01	0.72, 1.42
High-density lipoprotein cholesterol (per 10 mg/dl)	0.84	0.75, 0.93

Black race was an independent predictor of a subsequent coronary event. Other independent predictors of a coronary event included left ventricular hypertrophy, history of diabetes mellitus, number of calcified coronary arteries, history of hypertension and total cholesterol levels. High-density lipoprotein cholesterol was a significant independent and inverse predictor of a coronary event.

There were a total of 126 deaths in the entire cohort (11 [11.8%] blacks and 115 [9.0%] whites; p = 0.35). The majority (85 [67.5%]) of deaths were not attributed to CHD by the Adjudication Committee. In all instances where the Adjudication Committee decided a death was due to CHD, the decision was based upon the occurrence of either a sudden unexpected death or an MI death. There were no autopsy-determined CHD deaths.

There were more MIs (either fatal or nonfatal) in black subjects compared to white subjects (9 blacks [9.7%]; 62 whites [4.8%]; p = 0.05). Black subjects were also more likely to suffer new onset angina (14 blacks [15.1%]; 97 whites [7.6%]; p = 0.02). There was also a trend towards more hard events (CHD death or MI) in black subjects compared to whites, but the difference did not reach statistical significance (11 blacks [11.8%]; 86 whites [6.7%]; p = 0.09). The performance of revascularization procedures (percutaneous coronary angioplasty or coronary bypass) did not differ between the races (4 blacks [4.3%]; 76 whites [5.9%]; p = 0.65). There was also no significant ethnic difference in the occurrence of CHD deaths during the follow-up period (4 blacks [4.3%]; 37 whites [2.9%]; p = 0.35).

Table 6. Race, Calcification and Coronary End Points

	Events	No Events
Blacks		
Calcium present	10 (45.5%)	23 (32.4%)
Calcium absent	12 (54.5%)	48 (67.7%)
Whites		
Calcium present	138 (72.6%)	630 (57.7%)
Calcium absent	52 (27.4%)	462 (42.3%)

Numbers in parentheses indicate proportion of initial cohort for each race (blacks, n = 93; whites, n = 1,282).

Table 6 shows the relationship between calcification and end points (CHD death, MI, angina, or revascularization) in blacks and whites. Multivariate logistic regression analysis using the occurrence of any of these end points as the dependent variable revealed that black race was a significant and independent predictor of the occurrence of a CHD end point (OR 2.16; 95% CI 1.34, 3.48) (Table 7). Additional significant and independent predictors of a CHD event included the presence of LVH by ECG (OR 2.18; 95% CI 1.35, 3.52), a history of diabetes mellitus (OR 1.21; 95% CI 1.02, 1.43;), the number of calcified coronary vessels (OR 1.16; 95% CI 1.01, 1.33;), a history of hypertension (OR 1.34; 95% CI 1.02, 1.76) and total serum cholesterol (OR 1.03; 95% CI 1.00, 1.06). High-density lipoprotein cholesterol was a significant inverse predictor of a coronary endpoint (OR 0.84; 95% CI 0.75, 0.93). Age, history of smoking, gender and family history of CHD were not significant independent predictors of a CHD event.

DISCUSSION

Coronary calcium indicates concomitant atherosclerotic disease (31), and prospective studies have demonstrated that subjects with coronary calcium have a higher risk of subsequent coronary events compared to similar subjects without coronary calcium (19–24). However, we report here that in a cohort of asymptomatic subjects with coronary risk factors, coronary calcium detected with digital fluoroscopy was less prevalent in blacks than in whites, yet more blacks than whites suffered coronary events during a 70 month follow-up period. These results indicate that there are differences in a measurable pathobiologic characteristic of coronary lesions—calcific deposits—detected in vivo, in

two ethnic groups destined to suffer similar numbers of adverse outcomes.

Potential Explanations for Ethnic Differences

1. Differences in prevalence of atherosclerosis. It is possible that our black subjects had a lesser prevalence of coronary calcium because they had less coronary atherosclerosis than our white subjects. There are conflicting data on the prevalence and severity of coronary atherosclerosis in blacks and whites. Autopsy studies prior to the 1980s (32,33) have shown that blacks have less severe coronary atherosclerosis than whites, but more recent post-mortem data indicate that these differences are not significant (34). However, pathologic studies also have shown that blacks have less atherosclerotic calcium than whites (35,36). For example, Strong and McGill have reported that aortic lesions in whites were calcified 1.6 times more frequently than similar lesions in blacks (36). Consistent with these findings, our data also suggest that blacks have a lower frequency and severity of coronary calcium, despite similar or even greater risk of CHD events.

The National Institutes of Health has planned a large, multiethnic investigation to determine the interaction of coronary calcification and ethnicity in the prediction of CHD events (37). This study should more fully define the relationship of calcium quantity to CHD events in several ethnic groups.

2. Differences in calcium metabolism. Ethnic differences in bone metabolism have been previously reported; for example, despite a lower calcium intake (38), blacks have greater bone mass (39) and a lower incidence of osteoporosis and hip fractures (40) compared to whites. Several studies (41–43) have demonstrated racial differences in endocrine factors that affect bone metabolism and that, given the similarities between atherosclerotic calcification and osteogenesis (44,45), could affect mineralization at lesion sites. We have recently shown that serum levels of $1\alpha,25(OH)_2D_3$ are inversely related to calcium mass (16,18), that blacks have higher levels of $1,25(OH)_2D_3$, but these differences are not sufficient to fully account for the observed racial differences in coronary calcium (16). It is therefore conceivable that ethnic differences in endocrine mechanisms in addition to vitamin D metabolism may be contributing factors. However, these possibilities, while perhaps explaining racial differences in the amount of coronary calcium, do not explain why blacks have more events than whites.

3. Myocardial Mass. Recent studies suggest that myocardial mass may be a more important factor leading to sudden cardiac death in blacks than in whites (15,46). This relationship might be pertinent to our findings. Although both a history of hypertension and LVH by ECG were significant independent predictors of events in the entire cohort, we did not find a more significant independent association between LVH (by ECG) and coronary events in blacks, perhaps due to inadequate power in our study.

4. Differing importance of risk factors. Population-based epidemiologic studies indicate that predictors of CHD mortality were similar in blacks and whites except that cigarette smoking carried a greater risk for blacks, while serum cholesterol carried a greater risk in whites (1,9). Data from the Meharry-Hopkins Study which prospectively evaluated risk factors in black and white physicians suggest that systolic blood pressure is a relatively more important predictor of CHD events in black compared to white physicians, but serum cholesterol is more important in white physicians (15). Although Framingham risk in our black and white cohorts was identical, risk factor differences (Table 1 and Results) could have accounted for some of the observed differences in CHD events.

Blacks with symptomatic CHD have a poorer prognosis compared to whites (3,47,48), which at least in part appears to be due to limited access of blacks to care (49) and decreased utilization of revascularization procedures by blacks (4,6,50,51). Since our subjects were asymptomatic and had no evidence of coronary disease at enrollment and since rates of revascularization were similar, any effects of ethnic differences in access to care on our results would probably have been minimal, but cannot be excluded.

Study limitations. Though ours was a population based study of the South Bay suburbs of Los Angeles, there is the possibility that differing referral patterns between whites and blacks might have influenced the results. Education and socioeconomic status have been shown to have an effect on CHD outcomes. These were not controlled for in our study. Our study has limited power for determining the predictive accuracy of coronary calcium in blacks. There were few black women in our sample and for this and other reasons, our results may not be applicable to other populations. Therefore, despite the fact that this was not significant in our cohort (Table 6), larger multi-ethnic sample sizes may clarify these issues (37).

Conclusion. Our results demonstrate that black subjects with coronary risk factors have a lower prevalence of coronary calcium than similar white subjects, but have at least as many CHD events on follow-up. Our black sample was too small to determine if coronary calcium is predictive of outcomes in blacks. Further investigations into ethnic differences in the mechanistic determinants of coronary heart disease events are needed and are underway. It is hoped that such data may result in improved treatment to minimize the morbidity and mortality of CHD, particularly in blacks.

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